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Thank you

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The Use of Lysostaphin in Treatment of Staphylococcal Wound Infections

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STAPHYLOCOCCAL wound infections continue to plague physicians in spite of the development of the semisynthetic penicillinase resistant penicillin. The development of infections depends on a variety of factors including host resistance, strain pathogenicity and inoculum size.

Methods. Lysostaphin, a factor isolated in 1960 from a white colony staphylococcus, is lytic for virtually all strains of *S. aureus*. The material has a molecular weight of 32,000 and is composed of a peptidase and a hexosaminidase.

The value of Lysostaphin as a deterrent to staph wound infection was evaluated in 200 gram female guinea pigs in which paramedian incisions were inoculated with 3.7×10^8 organisms. The organisms were either phage type 52/52A or 80/81 penicillin resistant coagulase positive *S. aureus* cultured from ward patients with hospital acquired infections. This large inoculum was used because the guinea pig is hard to infect due to both natural resistance and sparse fatty subcutaneous tissue. Sterile procedures were used, making incisions down to fascia in iodine and alcohol prepped shaved abdomens. Continuous silk sutures for closure were employed.

Results. As has been noted by other investigators, the only significant criterion of wound infections is gross purulence. Positive cultures in the wounds opened after seven days did not predictably accompany established clinical infections.

After establishing the ability to infect the animals, wounds were irrigated with a 0.1 per cent solution of Lysostaphin after inoculation. Ninety per cent of controls and 15 per cent of treated animals developed wound infections.

To evaluate the mechanical effects of the irrigation, 20 animals received 3×10^8 organisms, with ten irrigated with Lysostaphin and ten irrigated with normal saline. Half of each group was sacrificed and cultured in seven days. All saline irrigated animals developed wound infections, while none of the Lysostaphin irrigated wounds became infected.

The remaining animals were followed for two weeks, during which the Lysostaphin irrigated group remained clinically free of infection, while all of the saline irrigated wounds were open and infected. The Lysostaphin group was then challenged with intravenous Lysostaphin, which produced anaphylactic deaths in all.

In attempting to determine if any of the effects were due to systemic effects of absorbed Lysostaphin, several technics were evaluated. All were judged by the presence or absence of clinical infection. If given SQ or intracardiac at the time of surgery, 50 per cent of controls and 22 per cent of treated became infected, but if administered at any time postoperatively there was no difference between the treatment and control groups.

Because of the anaphylaxis in the previously mentioned guinea pigs, this aspect was further studied in mice. All animals were sensitized to the drug IV or SQ and were then challenged by the same route three weeks later. All animals displayed an anaphylactin reaction with a 60 per cent mortality rate. In another group of animals it was shown that anaphylaxis required a minimum two week delay before challenge.